

Responses of Three Species of Captive Fruit-Eating Birds to Phosmet-Treated Food

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Abstract: We conducted two-cup feeding trials to assess the responses of cedar waxwings (*Bombycilla cedrorum* (Vieillet)), American robins (*Turdus migratorius* (L.)), and European starlings (*Sturnus vulgaris* L.) to food adulterated with phosmet 500 g kg⁻¹ WP ('Imidan'®). All species avoided phosmet concentrations ≥ 100 mg kg⁻¹. Robins avoided food containing 60 mg kg⁻¹ phosmet, while food consumption by starlings was reduced by a 30 mg kg⁻¹ treatment. All species were indifferent to phosmet at 10 mg kg⁻¹.

These findings suggest that fruit-eating birds will safely avoid ingesting harmful levels of phosmet when the insecticide is applied to small fruit such as blueberries and cherries. Feeding deterrence associated with phosmet might be exploited in management strategies to protect small fruit crops from damage by birds.

1 INTRODUCTION

Because of its toxicity to mammals (LD₅₀ for male laboratory rats = 113 mg kg⁻¹),¹ phosmet is considered a highly toxic pesticide.² There is, however, considerable variability in this compound's effect on wildlife. Among the few avian species tested, LD₅₀ values range from 18 mg kg⁻¹ for red-winged blackbirds (*Agelaius phoeniceus* (L.))³ to 1830 mg kg⁻¹ in mallards (*Anas platyrhynchos* L.).⁴

Furthermore, median lethal dietary concentrations range from 501 mg kg⁻¹ in northern bobwhite (*Colinus virginianus* (L.)) to > 5000 mg kg⁻¹ in mallards.⁵

Fruit-eating birds such as American robins (*Turdus migratorius* (L.)), cedar waxwings (*Bombycilla cedrorum* (Vieillet)), and European starlings (*Sturnus vulgaris* L.) are exposed to phosmet when it is applied for insect control to such crops as blueberries and grapes. Because fruit pulp is bulky and relatively unnutritious, frugivorous birds must consume substantial amounts each day to acquire adequate nourishment.⁶ Frugivorous species, then, would probably be among those receiving the highest dietary exposure to phosmet in the field. The extent to which fruit-eating birds can detect and avoid eating phosmet-treated food will affect their exposure to the pesticide.

Although there have been unsubstantiated reports that birds in the field are repelled by phosmet-treated fruit (Crites, W., 1991, pers. comm.), experiments specifically designed to examine this question have not been conducted. This is of interest not only from the standpoint of potential avian hazard but also because bird damage to small fruit is a major problem,^{7,8} and few effective control measures are available.^{9,10} Thus we conducted feeding trials with three common species of fruit-eating birds in order to evaluate their responses to food treated with phosmet.

2 METHODS

All birds were captured locally in Alachua County, Florida, under appropriate state and federal permits. We obtained starlings and cedar waxwings in February and April 1991, respectively, and tested both species in May 1991. We trapped and tested robins in February 1993. The birds were group-housed by species in 1.3 × 1.3 × 1.7 m cages in a covered outdoor aviary.

In captivity, the birds were given diets tailored to meet the individual species' needs. We initially maintained the waxwings on a banana mash diet.¹¹ Then, we gradually switched them to a maintenance diet of canary-finch feed

(AVN®, Purina Mills Inc., St. Louis, MO) supplemented 2–3 times weekly with fresh blueberries. We used the dry canary-finch feed because, unlike banana mash, it stayed fresh in the birds' cages. The starlings received game-bird starter (F-R-M®, Flint River Mills, Bainbridge, GA) exclusively until approximately two weeks prior to the feeding trial. At that time, we altered the starlings' regimen by giving them banana mash 3–4 h per day to familiarize them with the eventual test food. For the robins, we provided a maintenance diet of banana mash, mealworms (*Tenebrio* sp.), and fresh fruit.

One week prior to the start of each trial, we randomly assigned birds to treatment groups, weighed them, and placed them in individual test cages in another roofed outdoor aviary where they had continuous access to water and maintenance food.

On each of five days prior to testing we accustomed the birds to the test regime. We removed maintenance food at 0700, and 1 h later we gave each bird two plastic cups (8.2 cm diameter, 3.8 cm high) of banana mash, 30 g per cup for waxwings and starlings and 50 g per cup for robins. After 3 h, we removed the cups of mash and replaced the maintenance food.

Prior to the first day of the test, we randomly determined for each cage the locations of test cups A and B. These locations remained fixed for the duration of the test period. On Day 1, both cup A and B contained 30 g of untreated mash. On Days 2–5, cup A continued to hold untreated mash while cup B contained mash treated with phosmet. Thus, we assessed the birds' responses to treated food in relation to a single day of pretreatment consumption.¹²

Treatment levels for waxwings (four birds per group) were 10, 100 and 1000 mg phosmet kg⁻¹. We exposed starlings (five birds per group) to concentrations of 10, 30 and 100 mg kg⁻¹, and robins (four birds per group) to 10, 60, and 100 mg kg⁻¹. During the daily 3-h test we also put two cups of mash in an empty cage to determine evaporative water loss.

We tested cedar waxwings first, and we chose a broad range of test levels because the number of birds available for testing was limited and we were not certain what treatment rates would elicit a response. In subsequent tests, we retained 10 and 100 mg kg⁻¹ as lower and upper limits, and selected intermediate levels that approximated to probable residues in the field (Crites, W., unpublished).

We prepared each level of treated test food by combining the appropriate amount of phosmet 500 g kg⁻¹ WP ('Imidan®' 50-WP; Gowan Co., Yuma, AZ) with 1800 g of banana mash in a stainless steel mixing bowl. The treated food was prepared for each species immediately prior to its initial use on Day 2. The mash was kept refrigerated in the bowls, covered with aluminum foil, for use on subsequent days. The untreated mash was handled similarly. We did not determine phosmet residues.

After the daily 3-h test, we observed each bird briefly for clinical signs of intoxication, such as ataxia or

uncoordinated movements. We removed and weighed cups A and B, and after correcting for evaporative water loss, we determined consumption by subtraction. We evaluated consumption (g per cup) in a three-way repeated measures ANOVA, with phosmet level, day, and cup as main effects. Significant ($P < 0.05$) differences among means were isolated using Tukey's test.¹³

We estimated phosmet exposure in each group of test birds from the mean consumption of phosmet-treated food, the treatment rate, and the mean mass of the birds. Following the test on Day 5, we weighed and released each bird. We assessed the effect of treatment level on body mass using one-way ANOVAs.

3 RESULTS

3.1 Cedar waxwings

Each of the main factors and their interactions except cup \times level produced statistically significant effects on fruit mash consumption. Across all days, food consumption was higher ($P = 0.024$; $F = 5.8$; 2,9 df) in the 10 mg kg⁻¹ group ($\bar{x} = 13.4$ g per cup, S.E. = 1.3) than in both the 100 mg kg⁻¹ ($\bar{x} = 11.2$ g per cup, S.E. = 1.7) and 1000 mg kg⁻¹ ($\bar{x} = 10.1$ g per cup, S.E. = 1.7) groups. Across all treatment levels, consumption was lowest ($P = 0.001$; $F = 8.2$; 4,36 df) on Day 2, the initial treatment day ($\bar{x} = 9.2$ g per cup, S.E. = 1.8), than on Day 1, the pretreatment day ($\bar{x} = 11.6$ g per cup, S.E. = 1.8), or on the subsequent treatment days (12.2–12.4 g per cup). Across days and levels, waxwings consumed more ($P = 0.001$; $F = 100.4$; 1,9df) food from cup A ($\bar{x} = 18.4$ g, S.E. = 1.0) than from cup B ($\bar{x} = 4.7$ g; S.E. = 0.9).

The significant day \times level interaction ($P < 0.001$; $F = 5.6$; 8,36 df) reflected reduction in mash consumption by the 1000 mg kg⁻¹ treatment group on Day 2 compared to other days and levels. The significant day \times cup interaction ($P < 0.001$; $F = 18.4$; 4,36 df) indicated increasing consumption from cup A and decreasing consumption from cup B from Day 1 (pretreatment) through Days 2–4. The significant three-way interaction ($P < 0.01$; $F = 3.1$; 8,36 df) reflected that the divergence in cup use was most pronounced in the 100 and 1000 mg kg⁻¹ treatment groups (Fig. 1).

3.2 European starlings

Of the main factors, only day ($P < 0.001$; $F = 6.11$; 4,48 df) significantly affected food consumption. Across all groups, consumption of Day 5 ($\bar{x} = 12.4$ g per cup, S.E. = 2.3) exceeded that of the other four days (10.1–11.4 g per cup).

Although cup was not significant ($P = 0.705$; $F = 0.15$; 1,12 df) as a main effect, each interaction term involving cup did influence consumption. The cup \times level

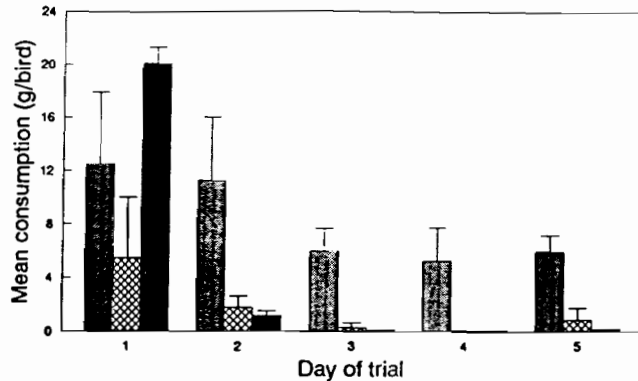


Fig. 1. Daily consumption of food from cup B by captive cedar waxwings (n = four birds per group) during five-day trials. On Day 1, all food was untreated; on Days 2–5 cup B held food treated with phosmet at (■) 10, (▨) 100, (■) 1000 mg kg⁻¹. Capped bars denote 1 S.E.

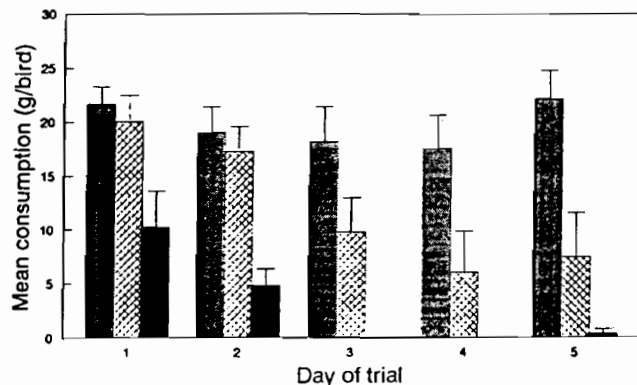


Fig. 2. Daily food consumption from cup B by captive European starlings (n = five birds per group) during five-day trials. On Day 1, all food was untreated; on Days 2–5, cup B held food treated with phosmet at (■) 10, (▨) 30, (■) 100 mg kg⁻¹. Capped bars denote 1 S.E.

interaction ($P = 0.02$; $F = 5.56$; 2,12 df) reflected greater use of cup A (untreated) by the 100 mg kg⁻¹ group than by the lower treatment levels. The day \times cup interaction ($P < 0.001$; $F = 8.79$; 4,48 df) indicated decreased use of cup B from Day 1 through Day 5. The three-way interaction ($P = 0.045$; $F = 2.19$; 8,48 df) reflected that the avoidance of cup B was most pronounced in the 100 mg kg⁻¹ group (Fig. 2).

3.3 American robins

Total food consumption (treated plus untreated) did not vary ($P = 0.722$; 2,9 df; $F = 0.34$) among groups. Across groups, consumption was higher ($P < 0.001$; $F = 9.85$; 4,36 df) on Day 1 (16.7 g per cup; S.E. = 0.9) than on the subsequent four treatment days (11.9–13.8 g per cup). Food consumption from cup B (8.4 g per bird; S.E. = 1.0) was lower ($P < 0.001$; $F = 20.44$; 1,9 df) than from cup A (18.5 g per bird; S.E. = 1.3).

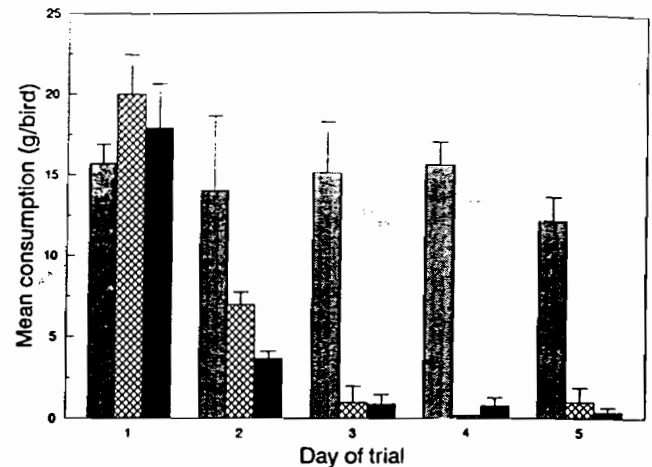


Fig. 3. Daily consumption from cup B by captive American robins (n = four birds per group) during five-day trials. Cup B held untreated food on Day 1; thereafter cup B held food treated with phosmet at (■) 10, (▨) 60, (■) 100 mg kg⁻¹. Capped bars denote 1 S.E.

The interaction between cup and treatment level ($P = 0.007$; $F = 8.99$; 2,9 df) reflected indifference to the treatment by the 10 mg kg⁻¹ group and the strong preference for cup A over treated cup B expressed by the 60 mg kg⁻¹ group (23.6 g per cup vs. 5.9 g per cup) and the 100 mg kg⁻¹ group (20.6 g per cup vs. 4.7 g per cup). The interaction between cup and day ($P < 0.001$; $F = 9.54$; 4,36 df) was due to the increasing disparity in consumption between cups as the trial progressed (Fig. 3). The three-way interaction ($P = 0.005$; $F = 3.42$; 8,36 df) reflected that the divergence in cup use occurred only in the two highest treatment levels.

3.4 Body mass

Changes in body mass did not differ among treatment levels in cedar waxwings ($P = 0.640$; $F = 0.47$; 2,10 df), starlings ($P = 0.706$; $F = 0.36$; 2,12 df), or robins ($P = 0.376$; $F = 1.09$; 2,9 df). Overall, waxwings lost an average of 1.6 g (S.E. = 1.2), or 4.4 % of initial mass, during the trial. Starlings averaged a 5.3-g loss (S.E. = 0.1), or 6.8 % of initial body mass. Robins averaged a 1.2-g gain (S.E. = 1.4) in body mass (1.7 %) during the test. All birds survived and were released.

3.5 Phosmet exposure

Generally, among the three species and various phosmet levels tested, the birds' estimated exposure to phosmet varied from 1.73 to 4.05 mg kg⁻¹ (Table 1). Even the cedar waxwing group given the 1000 mg kg⁻¹ phosmet treatment consumed far less phosmet than the median acute lethal dose (18 mg kg⁻¹) reported for the red-winged blackbird, a similar-sized passerine.³

TABLE 1
Estimated Exposure to Phosmet (mg Phosmet Ingested/kg Body Mass) in Three Species of Fruit-Eating Birds given Phosmet-Treated Food 3 h per day for Four Days at Various Treatment Rates

Species	Mass (g)	Phosmet treatment rate (mg kg ⁻¹)				
		10	30	60	100	1000
Waxwing	38.1	1.92 ^a	—	—	2.12	10.15
Starling	76.9	2.52	4.05	—	1.79	—
Robin	79.8	1.77	—	1.73	1.90	—

^a Median acute lethal dose for the red-winged blackbird (approximate body mass 60 g) is 18.0 mg kg⁻¹.³

4 DISCUSSION

Responses to phosmet-treated food were consistent across species in that all were deterred by concentrations of 100 mg kg⁻¹ or greater, and all were indifferent to the 10 mg kg⁻¹ level. We do now know the basis for the birds' discrimination and avoidance of phosmet-treated food. The compound has a strong, unpleasant odor that we detected readily, even at the lower concentrations. Cedar waxwings and starlings are sensitive to various volatile substances,^{14,15} and odor may have played a part in the birds' feeding responses.

Additionally, the birds may have experienced post-ingestional discomfort from eating the treated food. Phosmet is an organophosphorus compound and as such probably inhibited cholinesterase activity when ingested.² The birds then may have associated the resultant discomfort with the odor, taste, or location of the treated food and avoided it thereafter. After each day's feeding trial, we observed no sign of ill effects in the test birds. Many birds lost body mass, but not in a pattern suggestive of treatment effects.

The levels we tested were considerably less than the lethal dietary concentrations determined for most other avian species.^{2,16} Thus, in our feeding trials, waxwings, starlings, and robins recognized and learned to avoid phosmet-treated food at dietary concentrations well below lethal levels. The estimated amounts they ingested (Table 1) did not approach the lowest median acute lethal dose known for birds.^{2,3} We conclude that proper use of phosmet is not likely to endanger birds feeding on treated fruit.

The allowable phosmet residues on fruit at harvest range from 5 to 10 mg kg⁻¹, although concentrations on fruit soon after application may be 20–60 mg kg⁻¹ or more (Crites, W., unpublished). Thus, when applied to fruit, phosmet may have an initial repellent effect that could be exploited in the management of birds that cause millions of dollars worth of damage annually to crops such as grapes, cherries, and blueberries.^{7,8} This possibility needs to be evaluated further, particularly given the

current lack of alternative repellent materials for bird damage control.^{9,11}

Nevertheless, although our findings are encouraging, birds foraging on treated fruit in the field may not react as did the test subjects. The species we tested usually forage in large flocks. Through social facilitation, large numbers of actively foraging birds may influence individuals to persist in feeding on the fruit even though it is treated. Conceivably, the feeding activity of flockmates could induce individuals that have already learned to avoid phosmet-treated fruits to resume eating it.^{18,19} Also, birds may persist in eating phosmet-treated fruit if no ready alternative is available.

Further evaluation of phosmet as a bird repellent will help resolve these uncertainties. Singly caged birds exposed to food containing 20–80 mg kg⁻¹ phosmet should be tested in the absence of alternative food, and groups of birds should be tested in larger enclosures to assess the importance of social interactions. Pending the outcomes of these trials, field evaluation may be warranted.

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REFERENCES

1. Gaines, T. B., *Toxicol. Appl. Pharmacol.*, **14** (1969) 515–34.
2. Smith, G. J., *Pesticide use and toxicology in relation to wildlife: organophosphorus and carbamate compounds*. US Fish Wildl. Serv. Resource. Publ. 170, 1987, 171 pp.
3. Schafer, E. W., *Toxicol. Appl. Pharmacol.*, **21** (1972) 315–30.
4. Hudson, R. H., Tucker, R. K. & Haegele, M. A., *Handbook of toxicity of pesticides to wildlife*. 2nd edn. US Fish Wildl. Serv. Resour. Publ. 153, 1984, 90 pp.

5. Hill, E. F., Heath, R. G., Spann, J. W. & Williams, J. D., *Lethal dietary toxicities of environmental pollutants to birds*. US Fish Wild. Serv. Spec. Sci. Rep.—Wildl. 191, 1985, 61 pp.
6. Snow, B. & Snow, D., *Birds and Berries*. T & A D Poyser Ltd, Calton Waterhouses, Staffordshire, 1988, 268 pp.
7. Avery, M. L., Nelson, J. W. & Cone, M. A., *Proc. East. Wildl. Damage Control Conf.*, 5 (1991) 105–10.
8. Brown, R. G., *Canad. Wildl. Serv. Rep.*, 17 (1974) 1–57.
9. Tobin, M. E., & Dolbeer, R. A., *Proc. East. Wildl. Damage Control Conf.*, 3 (1987) 149–58.
10. Tobin, M. E., Woronecki, P. P., Dolbeer, R. A. & Bruggers, R. L., *Wildl. Soc. Bull.*, 16 (1988) 300–3.
11. Denslow, J. S., Levey, D. J., Moermond, T. C. & Wentworth, B. C., *Wilson Bull.*, 99 (1987) 131–4.
12. Mason, J. R., Avery, M. L., Glahn, J. F., Otis, D. L., Matteson, R. E. Nelms, C. O., *J. Wildl. Manage.*, 55 (1991) 182–7.
13. Steel, R. G. D. & Torrie, J. H., *Principles and procedures of statistics*, 2nd edn. McGraw-Hill Book Co., New York, 1980, 633 pp.
14. Clark, L., *Auk*, 108 (1991) 177–80.
15. Clark, L. & Mason, J. R., *Anim. Behav.*, 35 (1987) 227–35.
16. Hill, E. F. & Camardese, M. B., *Lethal dietary toxicities of environmental contaminants and pesticides to Coturnix*. US Fish Wildl. Serv. Fish Wildl. Tech. Rep. 2, 1986.
17. Avery, M. L., Cummings, J. L., Decker, D. G., Johnson, J. W., Wise, J. C. & Howard, J. I., *Crop Protect.*, 12 (1993) 95–100.
18. Klopfer, P. H., *Behaviour*, 14 (1959) 282–99.
19. Turner, E. R. A., *Behaviour*, 24 (1964) 1–46.